

## WEST Search History

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DATE: Wednesday, March 10, 2004

Hide?	<u>Set</u> <u>Name</u>	<u>Query</u>	<u>Hit</u> <u>Count</u>
		<i>DB=PGPB,USPT,EPAB,JPAB,DWPI; PLUR=YES; OP=OR</i>	
<input type="checkbox"/>	L9	L8 and l7	95
<input type="checkbox"/>	L8	l2 and reactivat\$ same (HSV\$3 or herpes adj simplex)	304
<input type="checkbox"/>	L7	L6 not l1	132
<input type="checkbox"/>	L6	L5 and (inhibit\$ or prevent\$ or treat\$ or reduc\$) same (reactivat\$5)	133
<input type="checkbox"/>	L5	(HSV\$3 or herpes adj simplex) and reactivat\$5 same (model or mouse)	199
<input type="checkbox"/>	L4	L3 and (scratch or abasion or dermabrasion) same (mouse rabbit animal monkey model)	2
<input type="checkbox"/>	L3	L2 not l1	900
<input type="checkbox"/>	L2	(HSV\$3 or herpes adj simplex) and reactivat\$5 and (model or mouse)	904
<input type="checkbox"/>	L1	(mouse rabbit animal monkey model) same (scratch \$4abras\$4) same (herpes or HSV\$3)	35

END OF SEARCH HISTORY

# STN Search History

FILE 'HOME' ENTERED AT 15:10:46 ON 10 MAR 2004

L1 QUE (HSV## OR HERPES (A) SIMPLEX) (P) (REACTIVAT##### OR RECRUD#####)  
L4 456 L3 AND (UV OR ULTRA-VIOLET OR ULTRVIOLET OR RADIAT#### OR IRRADIAT#####)  
L6 518 L1 AND (HSV## OR HERPES) (S) (UV OR ULTRA-VIOLET OR ULTRVIOLET OR RADIAT#### OR IRRADIAT#####)  
L9 523 L2 AND (UV OR ULTRA-VIOLET OR ULTRVIOLET OR RADIAT#### OR IRRADIAT#####) (S) (REACTIVAT##### OR RECRUD#####)

(FILE 'HOME' ENTERED AT 15:10:46 ON 10 MAR 2004)

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, AQUASCI, BIOBUSINESS, BIOCOMMERCE, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DISSABS, DDFB, DDFU, DGENE, DRUGB, DRUGMONOG2, ...' ENTERED AT 15:11:11 ON 10 MAR 2004

L1 QUE (HSV## OR HERPES (A) SIMPLEX) (P) (REACTIVAT##### OR RECRUD

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, LIFESCI, SCISEARCH' ENTERED AT 15:13:32 ON 10 MAR 2004

L2 6038 S L1  
L3 3800 S L2 AND (MOUSE OR MURINE OR ANIMAL OR MODEL OR RABBIT)  
L4 456 S L3 AND (UV OR ULTRA-VIOLET OR ULTRVIOLET OR RADIAT#### OR IR  
L5 204 DUP REM L4 (252 DUPLICATES REMOVED)  
L6 518 S L1 AND (HSV## OR HERPES) (S) (UV OR ULTRA-VIOLET OR ULTRVIOLE  
L7 149 S L6 AND L5  
L8 131 S L7 NOT PY>1999  
L9 523 S L2 AND (UV OR ULTRA-VIOLET OR ULTRVIOLET OR RADIAT#### OR IR  
L10 115 S L9 AND L8  
L11 115 DUP REM L10 (0 DUPLICATES REMOVED)  
L12 0 S L11 AND (SCRATCH OR ABRAS#####)  
L13 6 S L2 AND (SCRATCH OR ABRAS#####)  
L14 105 S L10 AND (HSV## OR HERPES ) (S) (REACTIVAT#####)  
L15 80 S L14 NOT PY>1990  
L16 35 S L10 NOT L15  
L17 80 S L15 NOT L16  
L18 62 S L15 AND (HSV## OR HERPES)/AB  
L19 43 S L15 AND (HSV## OR HERPES)/TI

L16 ANSWER 3 OF 35 MEDLINE on STN  
 AN 1999138953 MEDLINE  
 DN PubMed ID: 9971753  
 TI **Reactivation of herpes simplex virus type 1**  
 in the **mouse** trigeminal ganglion: an in vivo study of virus  
 antigen and cytokines.  
 AU Shimmeld C; Easty D L; Hill T J  
 CS Departments of Ophthalmology, University of Bristol, Bristol BS8 1TD,  
 United Kingdom.. C.Shimmeld@bris.ac.uk  
 SO Journal of virology, (1999 Mar) 73 (3) 1767-73.  
 Journal code: 0113724. ISSN: 0022-538X.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 199903  
 ED Entered STN: 19990316  
 Last Updated on STN: 19990316  
 Entered Medline: 19990304  
 AB **Reactivation of herpes simplex virus type 1**  
 (HSV-1) in the trigeminal ganglion (TG) was induced by  
**UV irradiation** of the corneas of latently infected  
**mice**. Immunocytochemistry was used to monitor the dynamics of  
 cytokine (interleukin-2 [IL-2], IL-4, IL-6, IL-10, gamma interferon  
 [IFN-gamma], and tumor necrosis factor alpha [TNF-alpha]) and viral  
 antigen production in the TG and the adjacent central nervous system on  
 days 1 to 4, 6, 7, and 10 after **irradiation**. **UV**  
**irradiation** induced increased expression of IL-6 and TNF-alpha  
 from satellite cells in uninfected TG. In latently infected TG, prior to  
**reactivation**, all satellite cells were TNF-alpha+ and most were  
 also IL-6(+). **Reactivation**, evidenced by HSV-1  
 antigens and/or infiltrating immune cells, occurred in 28 of 45 (62%) TG  
 samples. Viral antigens were present in the TG in neurons, often  
 disintegrating on days 2 to 6 after **irradiation**. Infected  
 neurons were usually surrounded by satellite cells and the foci of immune  
 cells producing TNF-alpha and/or IL-6. IL-4(+) cells were detected as  
 early as day 3 and were more numerous by day 10 (a very few IL-2(+) and/or  
 IFN-gamma+ cells were seen at this time). No IL-10 was detected at any  
 time. Our observations indicate that **UV irradiation**  
 of the cornea may modulate cytokine production by satellite cells. We  
 confirm that neurons are the site of **reactivation** and that they  
 probably do not survive this event. The predominance of TNF-alpha and  
 IL-6 following **reactivation** parallels primary infection in the  
 TG and suggests a role in viral clearance. The presence of Th2-type  
 cytokines (IL-4 and IL-6) indicates a role for antibody. Thus, several  
 clearance mechanisms may be at work.

L16 ANSWER 6 OF 35 MEDLINE on STN  
 AN 97042294 MEDLINE  
 DN PubMed ID: 8887494  
 TI **Reactivation of herpes simplex virus type 1**  
 in the **mouse** trigeminal ganglion: an in vivo study of virus  
 antigen and immune cell infiltration.  
 CM Erratum in: J Gen Virol 1996 Dec;77(Pt 12):3165  
 AU Shimmeld C; Whiteland J L; Williams N A; Easty D L; Hill T J  
 CS Department of Ophthalmology, School of Medical Sciences, Bristol, UK..  
 C.Shimmeld@bris.ac.uk  
 SO Journal of general virology, (1996 Oct) 77 ( Pt 10) 2583-90.  
 Journal code: 0077340. ISSN: 0022-1317.  
 CY ENGLAND: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 199612  
 ED Entered STN: 19970128  
 Last Updated on STN: 19980206  
 Entered Medline: 19961213

AB The corneas of latently infected **mice** were **UV irradiated** to induce **reactivation** of **herpes simplex** virus type 1 (**HSV-1**) in the trigeminal ganglion (TG). On days 1 to 4 after **irradiation**, TG were removed, serially sectioned and double stained to identify immune cells and virus antigens. Virus antigen was detected in small numbers (most commonly one) of neurons per ganglion as early as day 1, confirming the rapidity of **reactivation** and the neuron as the likely site of this event. The immune response was also rapid and effective since virus antigen was identified in immune cells at day 1 and by day 4 all samples were negative. The predominant infiltrating cells on days 1 and 2, when virus antigen was present and being cleared, were T cells, both CD4+ and CD8+. Later, large numbers of B cells appeared, suggesting that local antibody production may also be involved in controlling the **reactivated** infection. The observations suggest that a significant proportion of **reactivation** events do not result in disease of the eye or shedding of virus in the tear film. However, they also suggest that as little as one **reactivating** neuron in the ganglion may be sufficient to lead to such disease and/or shedding.

L16 ANSWER 8 OF 35 MEDLINE on STN  
 AN 96336924 MEDLINE  
 DN PubMed ID: 8760562  
 TI **UV radiation** and **mouse models** of **herpes** simplex virus infection.  
 AU Norval M; el-Ghorr A A  
 CS Department of Medical Microbiology, University of Edinburgh Medical School, Scotland.. M.Norval@ed.ac.uk  
 SO Photochemistry and photobiology, (1996 Aug) 64 (2) 242-5. Ref: 26  
 Journal code: 0376425. ISSN: 0031-8655.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 General Review; (REVIEW)  
 (REVIEW, TUTORIAL)  
 LA English  
 FS Priority Journals  
 EM 199610  
 ED Entered STN: 19961015  
 Last Updated on STN: 19961015  
 Entered Medline: 19961001

AB Orolabial human infections with **herpes simplex** virus type 1 (**HSV-1**) are very common; following the primary epidermal infection, the virus is retained in a latent form in the trigeminal ganglia from where it can **reactivate** and cause a **recrudescent** lesion. Recrudescences are triggered by various stimuli including exposure to sunlight. In this review three categories of **mouse models** are used to examine the effects of **UV irradiation** on **HSV** infections: these are **UV** exposure prior to primary infection, **UV** exposure as a triggering event for **recrudescence** and **UV** exposure prior to challenge with virus in **mice** already immunized to **HSV**. In each of these **models** immunosuppression occurs, which is manifest, in some instances, in increased morbidity or an increased rate of **recrudescence**. Where known, the immunological

mechanisms involved in the **models** are summarized and their relevance to human infections considered.

- L16 ANSWER 10 OF 35 MEDLINE on STN  
AN 93389452 MEDLINE  
DN PubMed ID: 8397285  
TI Effect of indomethacin on ultraviolet **radiation**-induced recurrent **herpes** simplex virus disease in guinea-pigs.  
AU Bratcher D F; Harrison C J; Bourne N; Stanberry L R; Bernstein D I  
CS Division of Infectious Diseases, Children's Hospital Research Foundation, Cincinnati, Ohio 45229.  
NC AI 22667 (NIAID)  
AI 23482 (NIAID)  
AI 29687 (NIAID)  
SO Journal of general virology, (1993 Sep) 74 ( Pt 9) 1951-4.  
Journal code: 0077340. ISSN: 0022-1317.  
CY ENGLAND: United Kingdom  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals  
EM 199310  
ED Entered STN: 19931105  
Last Updated on STN: 19970203  
Entered Medline: 19931018  
AB Exposure to u.v. **radiation** increases the local level of prostaglandins which may play a role in u.v. **radiation**-induced **herpes simplex** virus (**HSV**) recurrences. We used the guinea-pig **model** of u.v. **radiation**-induced recurrent genital **HSV-2** disease for examining the effects of indomethacin, a prostaglandin inhibitor, on u.v.-induced recurrences. In the first experiment, performed 100 days after **HSV-2** inoculation, treatment with indomethacin for 5 days begun 24 h before u.v.-**irradiation** decreased the proportion of **animals** developing **HSV** disease recurrences from 11/13 (84.6%) to 2/13 (15.4%) ( $P < 0.001$ ). In the second experiment, performed 135 days after **HSV-2** inoculation, treatment with indomethacin for 5 days begun 24 h before u.v.-**irradiation** decreased the number of **animals** developing recurrences from 12/21 (57.1%) to 5/21 (23.8%) ( $P < 0.05$ ). Five days of indomethacin treatment begun 4 h after u.v.-**irradiation**, however, did not reduce the percentage of **animals** developing disease recurrences but did decrease the mean number of days with recurrent lesions in **animals** that developed recurrences. Our data suggest that indomethacin may modify u.v. **radiation**-induced recurrent lesions by decreasing viral **reactivation** when given before u.v. **radiation** exposure or by reducing prostaglandin-induced immunosuppression when given before or after exposure. Future studies are needed for evaluating indomethacin prophylaxis for recurrent **HSV** disease when prolonged u.v. **radiation** exposure is anticipated.
- L16 ANSWER 12 OF 35 MEDLINE on STN  
AN 92364375 MEDLINE  
DN PubMed ID: 1323616  
TI **UV** light-induced **reactivation** of **herpes simplex** virus type 2 and prevention by acyclovir.  
AU Rooney J F; Straus S E; Mannix M L; Wohlenberg C R; Banks S; Jagannath S; Brauer J E; Notkins A L  
CS Laboratory of Oral Medicine, National Institute of Dental Research, National Institutes of Health, Bethesda, MD 20892.  
SO Journal of infectious diseases, (1992 Sep) 166 (3) 500-6.  
Journal code: 0413675. ISSN: 0022-1899.

CY United States  
 DT (CLINICAL TRIAL)  
 (CONTROLLED CLINICAL TRIAL)  
 Journal; Article; (JOURNAL ARTICLE)  
 (RANDOMIZED CONTROLLED TRIAL)  
 LA English  
 FS Abridged Index Medicus Journals; Priority Journals  
 EM 199209  
 ED Entered STN: 19920925  
 Last Updated on STN: 19960129  
 Entered Medline: 19920917  
 AB **UV** B light is a potent stimulus for inducing **reactivation** of latent **herpes simplex** virus (**HSV**) infections. Patients were enrolled in a double-blind placebo-controlled crossover trial to determine whether acyclovir can prevent **UV** light-induced **HSV**-2 recurrences. Twenty-four patients with a history of recurrent infection of perigenital sites (e.g., buttock, thigh) were exposed one to four times with 4 minimum erythema doses of **UV** light. Patients were given acyclovir 200 mg orally five times daily or matched placebo beginning 1 day before each exposure and continuing for 5 days after exposure. There were 13 **UV**-induced recurrences among 36 placebo treatments and 3 after 38 acyclovir treatments ( $P = .004$ ). The mean time to recurrence ( $\pm$  SE) was  $4.8 \pm 0.3$  days. **HSV**-2 lesions developed primarily at the site of **UV** exposure. The cutaneous distribution and timing of **UV**-induced recurrences was consistent with a neural localization (dorsal root ganglia) of latent viral infection. This **UV** light **model** permits direct examination of events leading to **HSV**-2 recurrences in humans and can be used to evaluate approaches to prevention.

L16 ANSWER 14 OF 35 MEDLINE on STN  
 AN 91373115 MEDLINE  
 DN PubMed ID: 1654309  
 TI Characterization of a **murine model** of recurrent **herpes simplex** viral keratitis induced by ultraviolet B **radiation**.  
 AU Laycock K A; Lee S F; Brady R H; Pepose J S  
 CS Department of Ophthalmology and Visual Science, Washington University School of Medicine, St. Louis, Missouri 63110.  
 SO Investigative ophthalmology & visual science, (1991 Sep) 32 (10) 2741-6.  
 Journal code: 7703701. ISSN: 0146-0404.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 199110  
 ED Entered STN: 19911108  
 Last Updated on STN: 19911108  
 Entered Medline: 19911024  
 AB The authors characterized a **murine model** of **herpes simplex** virus (**HSV**) **reactivation** in which recurrent herpetic keratitis was obtained in up to 80% of **animals**. Five weeks after ganglionic latency was established in National Institutes of Health inbred **mice** after corneal inoculation, **HSV** type 1 (**HSV**-1) was **reactivated** by **irradiating** the previously inoculated eye with ultraviolet (**UV**) light. Comparison of different **UV** wavelengths showed UVB to be optimal for **reactivation**, with peak viral recurrence being induced by a total exposure of approximately 250 mJ/cm<sup>2</sup>. **Reactivated** infectious virus generally began to appear

in trigeminal ganglia 2 days postirradiation and was subsequently detectable in the cornea by both corneal swabbing and immunostaining for viral antigens. Two consecutive outbreaks of viral recurrence at the ocular surface were induced in selected **animals** by serial exposure to UVB. Advantages of this **model** over other **models** of recurrent keratitis are discussed.

L16 ANSWER 15 OF 35 MEDLINE on STN  
AN 88034956 MEDLINE  
DN PubMed ID: 2822847  
TI **A murine model of herpes simplex virus recrudescence.**  
AU Norval M; Howie S E; Ross J A; Maingay J P  
CS Department of Bacteriology, University of Edinburgh Medical School, U.K.  
SO Journal of general virology, (1987 Oct) 68 ( Pt 10) 2693-8.  
Journal code: 0077340. ISSN: 0022-1317.  
CY ENGLAND: United Kingdom  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals  
EM 198711  
ED Entered STN: 19900305  
Last Updated on STN: 19900305  
Entered Medline: 19871125  
AB **A murine model** is described in which **recrudescence of herpes simplex virus (HSV)** type 1 was achieved. C3H **mice** were shaved and **irradiated** with u.v. B light 3 days before being infected epidermally with a clinical isolate of **HSV**. Seven weeks or longer following the primary infection, the survivors were again shaved, **irradiated** with u.v. and mildly tape-stripped. **Recrudescent** lesions occurred in up to 80% of **mice** at the site of the original lesion in most cases, but also occasionally at other sites. Skin painting with u.v.-**irradiated** urocanic acid (a substance suggested to be a photomediator of the immunosuppressive effects of u.v.) in place of u.v.-**irradiation** induced some **recrudescence** but was not as efficient as u.v.-**irradiation**. Antibody titres to **HSV** had no value in predicting whether **recrudescence** would occur but lymphoproliferative responses in draining lymph nodes may provide some indication of viral activity at the epidermal site. A hypothesis is developed that u.v.-**irradiation** before primary infection with **HSV** induces a suppressive immune response to the virus which affects the virus-host interaction and accounts for a high incidence of **recrudescent** lesions on subsequent stimulus.

L16 ANSWER 16 OF 35 MEDLINE on STN  
AN 80187958 MEDLINE  
DN PubMed ID: 6246417  
TI Repair and mutagenesis of **herpes simplex virus** in **UV-irradiated** monkey cells.  
AU Lytle C D; Goddard J G; Lin C H  
SO Mutation research, (1980 Apr) 70 (2) 139-49.  
Journal code: 0400763. ISSN: 0027-5107.  
CY Netherlands  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals  
EM 198007  
ED Entered STN: 19900315  
Last Updated on STN: 19900315

Entered Medline: 19800726

AB Mutagenic repair in mammalian cells was investigated by determining the mutagenesis of **UV-irradiated** or unirradiated **herpes simplex** virus in **UV-irradiated** CV-1 monkey kidney cells. These results were compared with the results for **UV-enhanced virus reactivation** (UVER) in the same experimental situation. High and low multiplicities of infection were used to determine the effects of multiplicity **reactivation** (MR). UVER and MR were readily demonstrable and were approximately equal in amount in an infectious center assay. For this study, a forward-mutation assay was developed to detect virus mutants resistant to iododeoxycytidine (ICdR), probably an indication of the mutant virus being defective at its thymidine kinase locus. ICpR-resistant mutants did not have a growth advantage over wild-type virus in irradiated or unirradiated cells. Thus, higher fractions of mutant virus indicated greater mutagenesis during virus repair and/or replication. The data showed that: (1) unirradiated virus was mutated in unirradiated cells, providing a background level of mutagenesis; (2) unirradiated virus was mutated about 40% more in **irradiated** cells, indicating that virus replication (DNA synthesis?) became mutagenic as a result of cell **irradiation**; (3) **irradiated** virus was mutated much more (about 6-fold) than unirradiated virus, even in unirradiated cells; (4) cell **irradiation** did not change the mutagenesis of **irradiated** virus except at high multiplicity of infection. High multiplicity of infection did not lead to higher mutagenesis in unirradiated cells. Thus the data did not demonstrate UVER or MR alone to be either error-free or error-prone. When the two processes were present simultaneously, they were mutagenic.

L16 ANSWER 17 OF 35 CAPLUS COPYRIGHT 2004 ACS on STN

AN 1999:585614 CAPLUS

DN 131:346171

TI Treatment of **HSV-1** infection with immunoglobulin or acyclovir: comparison of their effects on viral spread, latency, and **reactivation**

AU LeBlanc, Rona A.; Pesnicak, Lesley; Godleski, Matthew; Straus, Stephen E.  
CS Medical Virology Section, Laboratory of Clinical Investigation, National Institutes of Health, Bethesda, MD, 20892-1888, USA

SO Virology (1999), 262(1), 230-236  
CODEN: VIRLAX; ISSN: 0042-6822

PB Academic Press

DT Journal

LA English

AB We compared Ig (IgG) and acyclovir (ACV) therapies on the establishment, maintenance, and **reactivation** from latency of **HSV-1** (McKrae) in a **mouse** ocular infection **model**. **Mice** were given one i.p. (IP) dose of human IgG 24 h after infection (Day 1 p.i.) or ACV in the drinking water from Days 1 to 7 p.i. Both treatments allowed similar percentages of **mice** to survive the infection and decreased ocular virus shedding as compared with untreated controls. At most time points, there were no differences between IgG- and ACV-treated **animals** with respect to tissue virus titers or in the rates of virus **reactivation** during explant cocultivation. However, after **UV** exposure, **HSV reactivated** in 30% of ACV-treated **mice** compared with 90% of IgG-treated **mice** (P = 0.02). Also by quant. PCR, we found more latent **HSV-1** DNA copies in IgG-treated **mice** compared with those given ACV (P = 0.02). IgG treatment protects **mice** from **HSV-1** infection essentially as well as ACV does. Nonetheless, it permits higher levels of latent infection and subsequent in vivo **reactivation**. These studies have



implications for the mechanism by which IgG functions to attenuate  
**HSV** infections and for its potential value as a therapeutic agent  
in humans. (c) 1999 Academic Press.

RE.CNT 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 29 OF 35 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
AN 1991:244618 BIOSIS  
DN PREV199140118783; BR40:118783  
TI NERVE GROWTH FACTOR DOES NOT SUPPRESS **UV**-B INDUCED  
**REACTIVATION** OF LATENT **HERPES SIMPLEX** VIRUS  
IN-VIVO.  
AU PEPOSE J S [Reprint author]; BRADY R H; LAYCOCK K A; OSBORNE P A; JOHNSON  
E M JR  
CS DEP OPHTHALMOL, WASH UNIV SCH MED, ST LOUIS, MO, USA  
SO Investigative Ophthalmology and Visual Science, (1991) Vol. 32, No. 4, pp.  
853.  
Meeting Info.: ANNUAL SPRING MEETING OF THE ASSOCIATION FOR RESEARCH IN  
VISION AND OPHTHALMOLOGY, SARASOTA, FLORIDA, USA, APRIL 28-MAY 3, 1991.  
INVEST OPHTHALMOL VISUAL SCI.  
CODEN: IOVSDA. ISSN: 0146-0404.  
DT Conference; (Meeting)  
FS BR  
LA ENGLISH  
ED Entered STN: 21 May 1991  
Last Updated on STN: 21 May 1991

L19 ANSWER 2 OF 43 MEDLINE on STN  
 AN 88307442 MEDLINE  
 DN PubMed ID: 2841857  
 TI Cutaneous **herpes** simplex virus lesions induced by ultraviolet **radiation**. A review of **model** systems and prophylactic therapy with oral acyclovir.  
 AU Spruance S L  
 CS Department of Medicine, University of Utah School, Salt Lake City, Utah 84132.  
 SO American journal of medicine, (1988 Aug 29) 85 (2A) 43-5. Ref: 25  
 Journal code: 0267200. ISSN: 0002-9343.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 General Review; (REVIEW)  
 (REVIEW, TUTORIAL)  
 LA English  
 FS Abridged Index Medicus Journals; Priority Journals  
 EM 198809  
 ED Entered STN: 19900308  
 Last Updated on STN: 19900308  
 Entered Medline: 19880922  
 AB **Animal** and human **models** of ultraviolet **radiation**-induced **herpes simplex** virus disease provide opportunities to study the mechanism of virus latency and **reactivation**. These **models** can also be used to study the efficacy of antiviral agents. Prophylactic oral acyclovir altered the development of ultraviolet **radiation**-induced **herpes** labialis under both natural and experimental conditions.

L19 ANSWER 7 OF 43 MEDLINE on STN  
 AN 83141569 MEDLINE  
 DN PubMed ID: 6298618  
 TI Enhanced survival of ultraviolet-**irradiated herpes** simplex virus in cells exposed to antiviral agents.  
 AU Schnipper L E; Lewin A A; Crumpacker C S  
 NC AI-52530 (NIAID)  
 NIA-1-P01-AG0059 (NIA)  
 SO Mutation research, (1983 Feb) 116 (2) 65-72.  
 Journal code: 0400763. ISSN: 0027-5107.  
 CY Netherlands  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 198304  
 ED Entered STN: 19900318  
 Last Updated on STN: 19970203  
 Entered Medline: 19830415  
 AB Enhanced survival of **UV-irradiated HSV-1** is demonstrated in monkey cells exposed to inhibitors of viral DNA synthesis. Phosphonoacetic acid (PAA), adenine arabinoside (ara-A), and cytosine arabinoside (ara-C) pretreatment of infected cells is associated with concentration-dependent **reactivation** of **UV-HSV** -1. At concentrations that result in enhanced virus survival, inhibition of cell DNA synthesis is observed by either ara-A or ara-C, but not by PAA. Pretreatment of uninfected cells with acycloguanosine (ACG) is not associated with **reactivation** of **irradiated HSV** -1, and this is probably due to insufficient generation of ACG-triphosphate, the active inhibitor of viral and cell DNA synthesis.

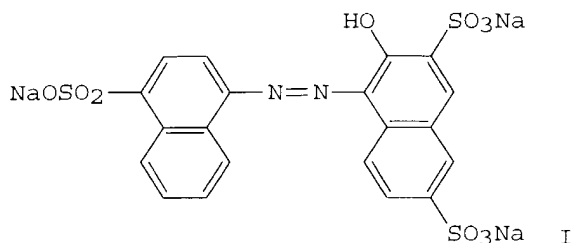
L19 ANSWER 11 OF 43 MEDLINE on STN

AN 80051198 MEDLINE  
 DN PubMed ID: 227991  
 TI Acute and latent infection of sensory ganglia with **herpes simplex** virus: immune control and virus **reactivation**.  
 AU Openshaw H; Asher L V; Wohlenberg C; Sekizawa T; Notkins A L  
 SO Journal of general virology, (1979 Jul) 44 (1) 205-15.  
 Journal code: 0077340. ISSN: 0022-1317.  
 CY ENGLAND: United Kingdom  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 198001  
 ED Entered STN: 19900315  
 Last Updated on STN: 19900315  
 Entered Medline: 19800124  
 AB The role of antiviral antibody in controlling the acute and latent phases of **herpes simplex** virus (HSV) infection in sensory ganglia of **mice** was studied in vitro and in vivo. Organ cultures of ganglia inoculated in vitro with HSV produced infectious virus for at least 3 weeks. In the presence of antiviral antibody, the titre of virus was markedly reduced, but the infection was not eliminated. Similarly, passive administration of antibody to HSV-infected immunodeficient (nude) **mice** reduced the virus titre but did not eliminate the acute phase of the ganglionic infection. Suppression of the cell-mediated immune response in latently infected immunocompetent **mice** by treatment with cyclophosphamide and/or X-**irradiation** resulted in **reactivation** of HSV in up to 70% of the **animals**. **Reactivation** was demonstrated by recovering infectious virus in cell-free homogenates of ganglia and eye globes and by finding virus antigens in ganglia by immunofluorescent staining. **Reactivation** occurred both in vitro and in vivo in the presence of high concentrations of neutralizing antibody. It is concluded that antibody alone is not sufficient to eliminate the acute phase of the ganglionic infection and that cytotoxic agents known to suppress the host's cellular immune response can **reactivate** virus in the presence of neutralizing antibody.

L19 ANSWER 13 OF 43 MEDLINE on STN  
 AN 74083359 MEDLINE  
 DN PubMed ID: 4359192  
 TI Multiplicity **reactivation** in UV-irradiated **herpes simplex** type 1 virus.  
 AU Roubal J; Vonka V  
 SO Intervirology, (1973) 1 (2) 73-9.  
 Journal code: 0364265. ISSN: 0300-5526.  
 CY Switzerland  
 DT Journal; Article; (JOURNAL ARTICLE)  
 LA English  
 FS Priority Journals  
 EM 197403  
 ED Entered STN: 19900310  
 Last Updated on STN: 19900310  
 Entered Medline: 19740320

L19 ANSWER 17 OF 43 CAPLUS COPYRIGHT 2004 ACS on STN  
 AN 1986:30038 CAPLUS  
 DN 104:30038  
 TI Influence of environmental substances on the **reactivation** of HSV-2 from latent infection systems in vitro  
 AU Fukuma, Mariko; Seto, Yoshiko; Toyoshima, Shigeshi  
 CS Sch. Med., Keio Univ., Tokyo, 160, Japan

SO Chemotherapy (Tokyo) (1985), 33(9), 733-42  
CODEN: NKRZAZ; ISSN: 0369-4682  
DT Journal  
LA Japanese  
GI



AB Amaranth (I) [915-67-3], erythrosine [16423-68-0], safrole [94-59-7], phenacetin [62-44-2], 2-acetylaminofluorene [53-96-3], SbCl<sub>3</sub>, and HgCl<sub>2</sub> inhibited the **reactivation** of **UV-irradiated herpes simplex** virus type 2 (**HSV-2**) from some latent infection system. Nicotine [54-11-5] enhanced the **reactivation** of **UV-irradiated HSV-2** at a nonpermissive temperature (NPT) to a permissive one, but it did not enhance the **reactivation** in an Ara C-treated cell system. None of the compds. **reactivated HSV-2** from the latent state at an NPT without any **reactivation**. Clear results could not be obtained in the trigeminal ganglion system because of a large variation of virus yield **reactivated**. Thus, some environmental substances may influence the onset and cure of recurrent **HSV** infection.

L19 ANSWER 31 OF 43 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

AN 1981:236262 BIOSIS

DN PREV198172021246; BA72:21246

TI LATENCY IN-VITRO USING **IRRADIATED HERPES SIMPLEX VIRUS**.

AU NISHIYAMA Y [Reprint author]; RAPP F

CS DEP MICROBIOL, PA STATE UNIV COLL MED, HERSHEY, PA 17033 USA, USA

SO Journal of General Virology, (1981) Vol. 52, No. 1, pp. 113-120.

CODEN: JGVIAIY. ISSN: 0022-1317.

DT Article

FS BA

LA ENGLISH

AB Human embryonic fibroblasts infected with **UV-irradiated herpes simplex** virus type 2 (**HSV-2**, strain 186) and maintained at 40.5° C did not yield detectable virus. Virus synthesis was induced by temperature shift-down to 36.5° C. The induced virus grew very poorly and was inactivated very rapidly at 40.5° C. Non-**irradiated** virus failed to establish latency at 40.5° C in infected cells. Enhanced **reactivation** of **HSV-2** was observed when latently infected cultures were superinfected with human cytomegalovirus (HCMV) or **irradiated** with a small dose of **UV** light at the time of temperature shift-down. HCMV did not enhance synthesis of **HSV-2** during a normal growth cycle but did enhance synthesis of **UV-irradiated HSV-2**. In this in vitro latency system, some **HSV** genomes damaged by **UV irradiation** were

maintained in a non-replicating state without being destroyed or significantly repaired.

- L19 ANSWER 34 OF 43 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN  
AN 1980:62471 BIOSIS  
DN PREV198018062471; BR18:62471  
TI ENHANCED **REACTIVATION OF UV IRRADIATED**  
**HERPES SIMPLEX VIRUS** INDUCED BY HUMAN CYTOMEGALOVIRUS.  
AU NISHIYAMA Y [Reprint author]; RAPP F  
CS PA STATE UNIV, COLL MED, HERSHEY, PA, USA  
SO Abstracts of the Annual Meeting of the American Society for Microbiology,  
(1980) Vol. 80, pp. ABSTRACT 954.  
Meeting Info.: 80TH ANNUAL MEETING, MIAMI BEACH, FLA., USA, MAY 11-16,  
1980. ABSTR ANNU MEET AM SOC MICROBIOL.  
CODEN: ASMACK. ISSN: 0094-8519.  
DT Conference; (Meeting)  
FS BR  
LA ENGLISH
- L19 ANSWER 42 OF 43 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.  
on STN  
AN 80084131 EMBASE  
DN 1980084131  
TI Enhanced survival of ultraviolet-**irradiated herpes**  
**simplex virus** in human cytomegalovirus-infected cells.  
AU Nishiyama Y.; Rapp F.  
CS Dept. Microbiol., Pennsylvania State Univ. Coll. Med., Hershey, Pa. 17033,  
United States  
SO Virology, (1980) 100/1 (189-193).  
CODEN: VIRLAX  
CY United States  
DT Journal  
FS 047 Virology  
LA English  
AB Infection of primary **rabbit** kidney cells and Vero cells with  
human cytomegalovirus (HCMV) enhanced survival of **uv-**  
**irradiated herpes simplex virus**. In primary  
**rabbit** kidney (PRK) cells, maximal enhancement was observed when  
cells were preinfected with HCMV at multiplicities of 1 to 10 PFU/cell and  
when HCMV-infected cells were used to assay for **uv-**  
**irradiated HSV** within 2 days postinfection.  
HCMV-enhanced **reactivation** was sensitive to **uv-**  
**irradiation**, although virus **reactivation** was much more  
resistant to **irradiation** than infectivity. Furthermore, the  
enhancement process was sensitive to caffeine and pretreatment of cells  
with cycloheximide augmented the enhancement induced by HCMV infection.
- L19 ANSWER 43 OF 43 LIFESCI COPYRIGHT 2004 CSA on STN  
AN 88:58834 LIFESCI  
TI Cutaneous **herpes simplex virus** lesions induced by ultraviolet  
**radiation**. A review of **model** systems and prophylactic  
therapy with oral acyclovir.  
WELLCOME INTERNATIONAL ANTIVIRAL SYMPOSIUM PROCEEDINGS.  
AU Spruance, S.L.; Lietman, P.S. [editor]; Fiddian, P. [editor]; Chapman,  
S.K. [editor]  
CS Div. Infect. Dis., Dep. Med., Univ. Utah Sch. Med., Salt Lake City, UT  
84132, USA  
SO AM. J. MED., (1988) pp. 43-45.  
Meeting Info.: Wellcome International Antiviral Symposium. Monte Carlo  
(Monaco). 2-4 Dec 1987.  
DT Book

TC Conference

FS V

LA English

SL English

AB **Animal** and human **models** of ultraviolet

**radiation**-induced **herpes simplex** virus disease

provide opportunities to study the mechanism of virus latency and

**reactivation**. These **models** can also be used to study the

efficacy of antiviral agents. Prophylactic oral acyclovir altered the

development of ultraviolet **radiation**-induced **herpes**

labialis under both natural and experimental conditions.